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Total α-Tocopherol Intakes Are Associated with Serum α-Tocopherol Concentrations in African American Adults[1,2]

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Abstract

African Americans in the southern United States have a high prevalence of chronic disease. Tocopherol intake and status have been associated with protection against several chronic diseases. Our objectives were, therefore, to examine the association between tocopherol intakes as measured by 2 regional FFQ and their corresponding concentrations in serum and to report on dietary sources of tocopherols in 404 men and women participating in the cross-sectional Diet and Physical Activity Sub-Study of the Jackson Heart Study. A large proportion (49% of men and 66% of women) reported dietary supplement use. Only 5.8% of men and 4.5% of women met the estimated average requirement (EAR) for vitamin E from foods alone, whereas 44.2% men and 49.2% women met it from foods and supplements. Total (diet + supplement) intake of α-tocopherol was associated with its corresponding measure in serum. Vitamin E supplement use, sex, serum cholesterol, education, and BMI, but not γ-tocopherol intakes, were associated with serum γ-tocopherol. For δ-tocopherol, associated variables included sex and serum cholesterol. The top food sources of α- and γ-tocopherol were snack chips and the top food source of δ-tocopherol was margarine. Despite prevalent vitamin E supplement use, more than one-half of this population did not meet the EAR for α-tocopherol intake and very few met it from food alone. Supplement use was associated with higher α- but lower γ-tocopherol concentration in serum. The possible health implications of this difference in relative tocopherol subtypes require further study. J. Nutr. 137: 2297–2303, 2007.

Introduction

Vitamin E has received considerable attention for its proposed role in the prevention of disease, including cardiovascular disease (1–3), cancer (4,5), cognitive decline (6,7), and poor immune function (8). However, clinical trials using vitamin E have produced inconsistent results (9). Meta analyses of studies with supplements (10,11) concluded that high-dose vitamin E supplements may actually be associated with increased mortality (11).

Vitamin E is a generic term that has generally referred to 4 different tocopherols and 4 tocotrienols (12). Although γ-tocopherol is the major form of vitamin E in the U.S. diet, α-tocopherol is the predominant form in serum due to its preferential incorporation by the hepatic α-tocopherol transfer protein.

In the US, health disparities are known to exist in ethnic and racial subsets of the population. One of the most striking cases of disparities is in cardiovascular health. African Americans have higher rates of morbidity and mortality from cardiovascular disease than the general population and the southeastern part of the US has the highest rates of hospitalization due to stroke and heart failure (13).

Given the health disparities that exist in African Americans with respect to cardiovascular disease and the protective role that tocopherol intake and status may play in prevention, the objectives of the current study were to: *1*) examine the dietary intake of tocopherols (α, γ, and δ) using 3 different dietary assessment instruments (2 region-specific FFQ and the mean of 4 24-h food recalls) and serum concentrations in African Americans participating in the Jackson Heart Study (JHS)[7]; *2*) evaluate associations between dietary intake measures and serum concentrations; and *3*) identify the relative contributions of foods to tocopherol intakes.

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[7] Abbreviations used: DPASS, Diet and Physical Activity Sub-Study; EAR, estimated average requirement; GED, General Educational Development; GLM, general linear model; JHS, Jackson Heart Study; LMD, Lower Mississippi Delta.

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Materials and Methods

Participants. Participants were from the JHS, a single-site prospective epidemiologic investigation of cardiovascular disease among African-Americans from the Jackson, Mississippi metropolitan area. JHS baseline data collection took place from the year 2000 to 2004. Data on conventional as well as new and emerging risk factors of cardiovascular disease were collected on a representative sample of African Americans, aged 34–84 y, residing in Hinds, Madison, and Rankin counties surrounding Jackson, Mississippi. A detailed description of the original study was published elsewhere (14).

Study sample selection. A subset of participants ($n = 499$) from the JHS cohort ($n = 5302$) were selected for the JHS Diet and Physical Activity Sub-Study (DPASS). The aim of DPASS was to provide data for validation of the diet and physical activity instruments used for the entire cohort of the JHS. The data presented here include all DPASS participants with complete dietary and serum tocopherol data. The Institutional Review Board of the University of Mississippi approved the DPASS protocols and all subjects gave written informed consent for their participation.

Dietary assessment. The most widely used FFQ were designed to capture foods most commonly consumed in the United States (15,16). This can lead to incorrect estimation of dietary intakes of population subgroups and ethnicities that have dietary practices that differ from the "general" population. These errors can lead to extensive misclassification.

The Lower Mississippi Delta (LMD) Nutrition Intervention Research Initiative, funded by the USDA Agricultural Research Service, conducted a telephone survey in the Delta region to collect representative dietary data using 24-h dietary recalls. These data were used to construct a new FFQ designed for use in the LMD region. Briefly, data from the survey were used to develop a regionally appropriate food list and portion size options for a FFQ and to provide weighting and recipe information for the nutrient database used to analyze questionnaire responses. Details regarding development of this regional FFQ are available elsewhere (17). This FFQ (283 items) and its shortened version (158 items), which was specifically developed for use in the JHS, were then used as dietary assessment tools in the DPASS. The short FFQ did not eliminate food groups but includes less detail by collapsing items for efficiency in reporting.

Four 24-h recalls were used to measure "actual" intake of DPASS participants. Dietary Intake data were collected using Nutrition Data System for Research software version 4.04 (2001) developed by the Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN. Recalls were conducted in person by registered dietitians who were trained and certified to use the software program. To ensure variability of intake, participants provided 2 weekday and 2 weekend days for the recalls.

The DPASS encounters were conducted in person and included an initial administration of the short FFQ, followed by 4 24-h interviewer-administered dietary recalls scheduled ~1 mo apart, and then administration of the long FFQ ~1 wk after administration of the last recall. Trained interviewers administered the recalls as well as the FFQ. Five percent of all FFQ and all the recalls were audio-taped for quality control purposes and were reviewed by the DPASS principal investigator. Retraining was conducted whenever problems with quality or completeness were identified by the review. Details regarding the methodology used for the DPASS were published elsewhere (18).

Laboratory analyses. Participants provided blood samples on the day of the baseline interview, which took place on the day of the administration of the short FFQ and, on average, 1 y prior to administration of the long FFQ. Blood samples from fasting (12 h) participants were collected in vacutainer tubes and centrifuged at 3000 × *g*; 10 min at 4°C. Serum was separated and frozen at −70°C until analyzed for tocopherols. Estimation of tocopherols was performed using HPLC as described previously (19). Serum cholesterol concentrations were determined according to methods described previously (20).

Covariate data. Nondietary information was obtained at the JHS baseline clinic visit. Age was computed from self-reported date of birth. Educational level was based on responses to questions on the highest grade or year of regular schooling and whether the participant had a high school diploma or General Educational Development (GED) certification. Smoking status was derived from a set of questions regarding tobacco use. Height and weight were both measured by trained technicians. Detailed procedures for anthropometric procedures have been detailed elsewhere (14). BMI was calculated as weight/height^2 (kg/m^2) from these measurements.

Statistical analysis. We excluded participants with reported energy intake outside the plausible range ≤ 2.51 or ≥ 16.73 MJ/d on any of the 3 dietary assessment methods ($n = 57$) or who had >10% of the questions blank on either of the 2 FFQ ($n = 5$). Participants without serum samples for antioxidant analysis ($n = 33$) were also excluded from analyses, leaving a sample of 404 individuals. For analyses with serum γ- and δ-tocopherol, we excluded 7 and 123 participants, respectively, with concentrations below detectable levels.

Both intake as well as serum levels of tocopherols were skewed and were log transformed prior to analyses. As a large proportion of participants took vitamin E supplements, we analyzed the data using both tocopherol intakes from diet as well as total intakes (diet + supplement).

Descriptive analyses were performed to assess sex differences using general linear models (GLM) and logistic regression for categorical data. Multiple linear regression analyses were performed to assess relationships between serum tocopherol measures with known dietary and nondietary correlates. For these analyses, tocopherol intakes were modeled as quartiles of intake. To observe the effect of taking vitamin E supplements on serum α-, γ-, and δ-tocopherol concentrations, an indicator variable (vitamin E supplement use, yes or no) was created and used in regression analyses. We also ranked the main food sources of tocopherols as reported on the long FFQ by calculating the percentage contribution of each food item to the total intake of each tocopherol. All α values were set at the 0.05 level and analyses were performed using SAS (release version 9.1, 2002–2003, SAS Institute).

Results

Mean age did not differ significantly between men and women (60 y vs. 62 y, range 35–80 y) (**Table 1**). Mean age-adjusted BMI

TABLE 1 Characteristics of male and female JHS-DPASS participants

Variable	Men, $n = 156$	Women, $n = 248$
Age, *y*	60.2 ± 0.76	61.5 ± 0.61
BMI[1], *kg/m^2* ($n = 403$)	29.4 ± 0.53	31.9 ± 0.42*
Smoking status, %		
Never	51.9	74.6
Former	35.3	19.0
Current	12.8	6.5*
Education,[2] % ($n = 401$)		
<12 y	20.1	16.2
High school diploma or GED	20.1	21.1
Vocational degree or some college	14.3	17.8
Associates/bachelor's/professional degree	45.5	44.9
Vitamin/mineral supplement users, %		
Short FFQ	46.8	62.9*
Long FFQ	50.6	68.2*

[1] Values are means ± SE. Sex groups were compared by GLM after adjusting for age.
*Different from men, $P < 0.05$.
[2] $n = 154$ (men) and 247 (women).

was higher for women than men (32 vs. 29 kg/m^2; $P < 0.05$). A lower percentage of women than men were current smokers ($P < 0.05$). Supplement use, as reported on both the FFQ, also varied by sex, with more women reporting use than men. Women had higher serum HDL and total cholesterol concentrations compared with men ($P < 0.05$) (Table 2). Serum LDL cholesterol and triglyceride concentrations did not differ. Women using supplements had significantly higher serum α-tocopherol than male supplement users. Women also had significantly higher serum γ-tocopherol concentrations compared with men (for both supplement users and nonusers). No sex differences were observed for δ-tocopherol concentrations in supplement users. However, in nonsupplement users, women had higher serum concentrations than men.

Women reported higher intakes of total α-tocopherol across all 3 dietary assessment methods (Table 3). Significantly higher intakes were also reported by women relative to men for γ-tocopherol on the long FFQ. Using dietary intake from the mean of the 4 24-h dietary recalls, 95.5% of women and 94.2% of men did not meet the estimated average requirement (EAR) of 12 mg/d (21) of α-tocopherol. When supplements were included, 55.8% of the women and 50.8% of men did not meet the EAR for α-tocopherol.

Regardless of dietary assessment method, total intake of α-tocopherol was strongly and positively associated with serum α-tocopherol ($P < 0.001$) (Table 4). Having completed high school or the GED ($P < 0.05$, except for short FFQ where $P < 0.1$), serum concentrations of total cholesterol ($P < 0.001$) and age ($P < 0.01$) were also positively associated with serum α-tocopherol. Energy intake was negatively associated with serum α-tocopherol for both the FFQ. A comparison of total and dietary intakes of α-tocopherol revealed that, in general, the highest 2 quartiles of total α-tocopherol intake represented supplement intake. When we examined dietary intake of α-tocopherol adjusted for vitamin E supplement use, dietary intake was no longer associated with serum measures except for the highest quartile of intake for the mean of the recalls (Table 5).

Except for the highest quartile as estimated by the short FFQ, intake of γ-tocopherol was not associated with its corresponding serum measure (Table 6). Nondietary factors associated with serum γ-tocopherol included serum total cholesterol, high school or GED completion (negatively), and BMI (all $P < 0.01$). Vitamin E supplement use was associated with lower

TABLE 3 Dietary intakes of tocopherols of JHS-DPASS participants by 3 methods[1,2]

Dietary nutrient	Mean of 4 24-h recalls	Short FFQ	Long FFQ
Men ($n = 156$)			
Energy, *MJ/d*	8.43 ± 0.17	8.61 ± 0.23	8.94 ± 0.22
Total α-tocopherol, *mg/d*	54.3 ± 10.6	91.0 ± 7.56	64.7 ± 9.26
% < EAR[3]	55.8	—	—
Dietary α-tocopherol, *mg/d*	6.11 ± 0.24	6.71 ± 0.17	6.76 ± 0.17
% < EAR[3]	94.2	—	—
γ-Tocopherol, *mg/d*	14.18 ± 0.44	13.09 ± 0.36	13.59 ± 0.37
δ-Tocopherol, *mg/d*	2.14 ± 0.09	2.45 ± 0.07	2.75 ± 0.07
Women ($n = 248$)			
Energy, *MJ/d*	6.92 ± 0.13*	7.48 ± 0.18*	7.60 ± 0.18*
Total α-tocopherol, *mg/d*	73.2 ± 8.3*	66.7 ± 9.58*	82.4 ± 7.29*
% < EAR[3]	50.8	—	—
Dietary α-tocopherol, *mg/d*	6.58 ± 0.19	6.86 ± 0.14	6.94 ± 0.13
% < EAR[3]	95.5	—	—
γ-Tocopherol, *mg/d*	14.60 ± 0.34	13.72 ± 0.28	15.25 ± 0.29*
δ-Tocopherol, *mg/d*	2.29 ± 0.07	2.43 ± 0.06	2.93 ± 0.06

[1] Values are means ± SE. *Different from men, $P < 0.05$.
[2] Pairwise comparisons using log-transformed variables, compared by GLM after adjusting for age and energy intake (from appropriate assessment tool) by sex.
[3] EAR for men and women = 12 mg α-tocopherol/d (21).

TABLE 2 Serum lipid and tocopherol concentrations (with and without supplement use) of male and female JHS-DPASS participants[1–3]

Serum measurement	Men	*n*	Women	*n*
LDL cholesterol, *mmol/L*	3.23 ± 0.07	154	3.19 ± 0.06	246
HDL cholesterol, *mmol/L*	1.21 ± 0.03	155	1.47 ± 0.02*	248
Total cholesterol, *mmol/L*	5.00 ± 0.08	156	5.25 ± 0.07*	248
Triglycerides, *mmol/ L*	1.25 ± 0.09	156	1.30 ± 0.07	248
α-Tocopherol, *μmol/ L*	28.52 ± 1.22	156	34.28 ± 0.97	248
Nonsupplement users	23.30 ± 0.84	83	25.10 ± 0.80	92
Supplement users	34.32 ± 2.01	73	39.75 ± 1.37*	156
α-Tocopherol:LDL cholesterol	9.35 ± 0.47	154	11.44 ± 0.37	246
Nonsupplement users	7.43 ± 0.27	81	7.98 ± 0.26	91
Supplement users	11.43 ± 0.78	73	13.50 ± 0.54*	155
γ-Tocopherol, *μmol/L*	4.98 ± 0.36	156	6.13 ± 0.29*	241
Nonsupplement users	6.26 ± 0.33	83	8.03 ± 0.32*	90
Supplement users	3.56 ± 0.60	73	4.99 ± 0.41*	151
δ-Tocopherol, *μmol/L*	0.34 ± 0.09	107	0.60 ± 0.07	174
Nonsupplement users	0.37 ± 0.11	67	0.62 ± 0.10*	82
Supplement users	0.30 ± 0.15	40	0.59 ± 0.10	92

[1] Values are means ± SEM. *Different from men, $P < 0.05$.
[2] Pairwise comparisons using log-transformed variables compared by GLM after adjusting for age by sex.
[3] Supplement use based on reported use of vitamin and/or mineral use on the short FFQ.

TABLE 4 Regression coefficients (β) and SE for associations of total α-tocopherol intake and other covariates with serum α-tocopherol concentration in the JHS-DPASS participants[1–3]

Dietary and nondietary predictors	Mean of 4 24-h recalls	Short FFQ	Long FFQ
Total intake, *mg/d*		$\beta \pm SE$	
Quartile 1 (Ref)	—	—	—
Quartile 2	0.05 ± 0.05	0.06 ± 0.04	0.17 ± 0.05***
Quartile 3	0.17 ± 0.05***	0.26 ± 0.04***	0.24 ± 0.05***
Quartile 4	0.50 ± 0.05***	0.58 ± 0.04***	0.51 ± 0.05***
Age (per 10 y)	0.07 ± 0.02***	0.05 ± 0.02**	0.06 ± 0.02***
Sex (female = 1)	0.07 ± 0.03*	0.04 ± 0.03	0.06 ± 0.03
Education (GED = 1)	0.12 ± 0.04**	0.07 ± 0.04	0.09 ± 0.04*
BMI, *kg/m^2*	0.03 ± 0.08	0.02 ± 0.07	0.01 ± 0.08
Serum cholesterol, *mmol/L*	0.73 ± 0.07***	0.76 ± 0.07***	0.72 ± 0.08***
Energy intake, *MJ/d*	−0.04 ± 0.06	−0.10 ± 0.04*	−0.13 ± 0.05**
R^2	0.43	0.51	0.41

[1] Serum concentrations and intake of tocopherol, energy intake, serum cholesterol concentrations and BMI were log transformed, $n = 401$. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.
[2] Values are β coefficients and SE.
[3] Median total α-tocopherol intakes for the quartile categories were 4.2, 7.7, 19.2, and 195 mg/d for the mean of the 4 recalls; 4.6, 7.6, 23.8, and 287 mg/d for the short FFQ; and 5.3, 8.0, 24.5, and 285 mg/d for the long FFQ.

TABLE 5 Regression coefficients (β) and SE for associations of dietary α-tocopherol intake and other covariates with serum α-tocopherol concentration in JHS-DPASS participants[1–3]

Dietary and nondietary predictors	Mean of 4 24-h recalls	Short FFQ	Long FFQ
Dietary intake, *mg/d*		$\beta \pm SE$	
Quartile 1 (Ref)	—	—	—
Quartile 2	0.04 ± 0.05	−0.01 ± 0.04	0.05 ± 0.05
Quartile 3	0.08 ± 0.05	0.07 ± 0.04	0.10 ± 0.05*
Quartile 4	0.16 ± 0.06**	−0.02 ± 0.04	0.05 ± 0.05
Vitamin E supplement use (yes = 1)	0.30 ± 0.03***	0.43 ± 0.03***	0.31 ± 0.03***
Age (per 10 y)	0.07 ± 0.02**	0.04 ± 0.02*	0.07 ± 0.02***
Sex (female = 1)	0.10 ± 0.04**	0.04 ± 0.03	0.06 ± 0.04
Education (GED = 1)	0.13 ± 0.04**	0.10 ± 0.04*	0.11 ± 0.05*
BMI, *kg/m²*	−0.03 ± 0.08	0.01 ± 0.07	−0.03 ± 0.08
Serum cholesterol, *mmol/L*	0.68 ± 0.08***	0.74 ± 0.07***	0.70 ± 0.08***
Energy intake, *MJ/d*	−0.12 ± 0.07	−0.07 ± 0.04	−0.10 ± 0.05*
R^2	0.37	0.49	0.36

[1] Serum concentrations and intake of tocopherol, energy intake, serum cholesterol concentrations, and BMI were log transformed, n = 401. *P < 0.05; **P < 0.01; ***P < 0.001.

[2] Values are β coefficients and SE.

[3] Median dietary α-tocopherol intakes for the quartile categories were 3.4, 5.0, 6.5, and 9.7 mg/d for the mean of the 4 recalls; 4.6, 5.6, 6.5, and 8.1 mg/d for the short FFQ; and 5.0, 5.8, 6.6, and 8.0 mg/d for the long FFQ.

TABLE 6 Regression coefficients (β) and SE for associations of γ-tocopherol intake and other covariates with serum γ-tocopherol concentration in JHS-DPASS participants[1–3]

Dietary and nondietary predictors	Mean of 4 24-h recalls	Short FFQ	Long FFQ
Dietary intake, *mg/d*		$\beta \pm SE$	
Quartile 1 (Ref)	—	—	—
Quartile 2	0.09 ± 0.09	0.09 ± 0.08	−0.01 ± 0.08
Quartile 3	0.10 ± 0.09	0.15 ± 0.08	0.09 ± 0.08
Quartile 4	0.08 ± 0.11	0.24 ± 0.08**	0.15 ± 0.06
Vitamin E supplement use (yes = 1)	−0.45 ± 0.06***	−0.68 ± 0.06**	−0.57 ± 0.06***
Age (per 10 y)	−0.07 ± 0.03*	−0.09 ± 0.03	−0.05 ± 0.03
Sex (female = 1)	0.07 ± 0.07	−0.17 ± 0.58**	−0.16 ± 0.06*
Education (GED = 1)	−0.23 ± 0.08**	−0.16 ± 0.07*	−0.21 ± 0.24*
BMI, *kg/m²*	0.50 ± 0.15**	0.49 ± 0.14***	0.55 ± 0.14***
Serum cholesterol, *mmol/L*	1.09 ± 0.14***	1.00 ± 0.13***	1.10 ± 0.14***
Energy intake, *MJ/d*	−0.15 ± 0.13	0.13 ± 0.07	0.25 ± 0.08**
R^2	0.30	0.43	0.38

[1] Serum concentrations and intake of tocopherol, energy intake, serum cholesterol concentrations, and BMI were log transformed, n = 394. *P < 0.05; **P < 0.01; ***P < 0.001.

[2] Values are β coefficients and SE.

[3] Median γ-tocopherol intakes for the quartile categories were 6.5, 12.0, 15.5, and 22.0 mg/d for the mean of the 4 recalls; 7.6, 10.5, 13.3, and 16.9 mg/d for the short FFQ; and 9.3, 12.2, 14.6, and 17.6 mg/d for the long FFQ.

serum γ-tocopherol concentrations (P < 0.01) and women had lower serum γ-tocopherol concentrations than men on the FFQ but not the recalls. For the mean of the recalls, age was also negatively associated with serum γ-tocopherol concentrations.

δ-Tocopherol intake was not associated with its serum measure (**Table 7**). Sex and serum cholesterol concentrations were positively associated with serum δ-tocopherol concentrations. Vitamin E supplement use (only for the short FFQ) was negatively associated with serum δ-tocopherol concentrations.

Dietary sources of tocopherols, as estimated by the long FFQ, were examined (**Table 8**). Snack chips, oils, and salad dressing and fish preparations were the top 3 contributors of α-tocopherol. Main sources of γ-tocopherol included snack chips, baked desserts, and corn preparations. The major sources of δ-tocopherol on the long FFQ included margarine, corn preparations, and baked desserts.

Discussion

Tocopherols have been associated both positively and negatively with chronic diseases (22). We assessed the intake and serum levels of tocopherols in the all-African American cohort of the JHS. Over 95% of our population did not meet the EAR for α- tocopherol from dietary sources.

Supplement use. Although not expected, we observed high supplement use. Forty nine percent of men and 66% of women (as reported on either of the FFQ) in our study reported taking supplements. As documented by various NHANES studies, there has been increased dietary supplement use in the United States (23). However, several studies have shown lower use among African Americans (24,25). Thus, we were surprised to see high usage in this population.

Serum levels of tocopherols. These southern-based African American adults had mean serum α-tocopherol concentrations comparable to nationally representative data (24,26). Supplement users in the DPASS of the JHS had a higher serum α-tocopherol concentration than nonusers. Serum γ-tocopherol concentrations in our study were similar to those seen in other populations (27–29).

Intake of tocopherols. The mean dietary intake of α-tocopherol, as measured by 3 dietary assessment tools, ranged from 6–8 mg/d. Data from the 1994–1996 Continuing Survey of Food Intakes by Individuals reported mean daily intakes of men in a similar range; however, dietary intakes of women were much lower than those reported by our study (30). Other studies using nationally representative data have estimated mean daily dietary vitamin E intakes in the range of 7–10 mg of α-tocopherol equivalents (21,31–33). The higher intakes reported in these studies could be attributed to differences in units used to express vitamin E intake. Our study estimated α-tocopherol intake as milligrams of α-tocopherol. However, until recently, α-tocopherol intake was estimated as α-tocopherol equivalents. Intake of α-tocopherol (in milligrams) is ~0.8 of total α-tocopherol equivalents (21).

Because of the ubiquitous presence of γ-tocopherol in vegetable oil, diets in the United States typically contain 2–4 times the amount of γ-tocopherol compared with α tocopherol (34). Intakes of γ-tocopherol in the participants of the JHS were similar to those reported by others (35).

Factors associated with serum tocopherol concentrations. Serum total cholesterol was associated with serum α-tocopherol in this group of adults. During absorption, α-tocopherol from the intestine is secreted in chylomicron particles along with

TABLE 7 Regression coefficients (β) and SE for associations of δ-tocopherol intake and other covariates with serum δ-tocopherol concentration in JHS-DPASS participants[1–3]

Dietary and nondietary predictors	Mean of 4 24-h recalls	Short FFQ	Long FFQ
Dietary intake, *mg/d*		$\beta \pm SE$	
Quartile 1 (Ref)	—	—	—
Quartile 2	−0.04 ± 0.11	−0.02 ± 0.11	0.13 ± 0.11
Quartile 3	0.02 ± 0.11	0.09 ± 0.11	0.07 ± 0.11
Quartile 4	0.21 ± 0.12	0.06 ± 0.11	0.14 ± 0.11
Vitamin E supplement use (yes = 1)	−0.09 ± 0.08	−0.21 ± 0.08**	−0.10 ± 0.08
Age (per 10 y)	−0.08 ± 0.04	−0.06 ± 0.04	−0.07 ± 0.04
Sex (female = 1)	0.18 ± 0.08*	0.23 ± 0.08**	0.22 ± 0.08**
Education (GED = 1)	−0.07 ± 0.10	−0.02 ± 0.10	−0.05 ± 0.10
BMI, *kg/m²*	−0.11 ± 0.19	−0.08 ± 0.19	−0.06 ± 0.19
Serum cholesterol, *mmol/L*	0.54 ± 0.19**	0.54 ± 0.19**	0.54 ± 0.19**
Energy intake, *MJ/d*	−0.11 ± 0.19	−0.10 ± 0.09	0.03 ± 0.11
R^2	0.09	0.10	0.08

[1] Serum concentrations of tocopherols, energy intake, serum cholesterol concentrations, and BMI were log transformed, $n = 281$. *$P < 0.05$; **$P < 0.01$.
[2] Values are β coefficients and SE.
[3] Median δ-tocopherol intakes in the quartile categories were 1.0, 1.7, 2.4, and 3.4 mg/d for the mean of the 4 recalls; 1.4, 2.0, 2.6, and 3.4 mg/d for the short FFQ; and 1.9, 2.5, 3.0, and 3.8 mg/d for the long FFQ.

TABLE 8 Main dietary contributors to tocopherol intakes among JHS-DPASS participants[1]

Nutrient	Contribution
α-Tocopherol	%
Snack chips	10.0
Oil and salad dressing	6.6
Fish preparations	6.4
Corn bread and muffins	6.2
Peanut and peanut preparations	5.2
Baked desserts	5.2
γ-Tocopherol	
Snack chips	13.8
Baked desserts	11.9
Corn bread and muffins	11.4
Oil and salad dressing	11.2
Fish preparations	6.6
Margarine	6.5
Potato and potato preparations	5.1
δ-Tocopherol	
Margarine	18.1
Corn bread and muffins	16.9
Baked desserts	14.8
White bread products	8.0
Rice preparations	7.3
Oil and salad dressing	7.0

[1] Those contributing at least 5% of dietary intake on the long FFQ are listed.

triacylglycerol and cholesterol. It is transported in blood as part of a lipoprotein complex and is therefore expected to be positively associated with lipids (27).

Serum α-tocopherol was associated with older age. However, this association did not persist when data were restricted to only nonsupplement users (data not shown), suggesting confounding due to supplement use. Investigators of the Women's Health Initiative have reported similar findings (29). Other studies have found no associations with age (36). Total α-tocopherol intake (diet + supplement), but not dietary intake, was associated with serum α-tocopherol concentrations. Low or no correlations between dietary intake and serum measures of α-tocopherol have been reported by Stryker et al. (37), Ascherio et al. (38), Brunner (39), El-Sohemy (40), Vogel et al. (41), and Dixon et al. (42).

In participants of the DPASS-JHS, one of the strongest predictors of serum γ-tocopherol was use of vitamin E supplements; supplement users had significantly lower serum γ-tocopherol. A decrease in serum γ-tocopherol following administration of vitamin E supplementation has been previously demonstrated (43). α and γ-Tocopherol have similar nonselective mode chylomicron-mediated absorption in the gut. However, in the liver, α-tocopherol is preferentially reincorporated into nascent VLDL by α-tocopherol transfer protein. α-Tocopherol supplements may reduce circulating γ-tocopherol concentrations due to competition for hepatic transfer, thereby leaving more γ-tocopherol available for degradation (44).

There was a positive association between BMI and γ-tocopherol concentrations. This has been reported previously in other studies (29,45,46). Food sources of γ-tocopherol in our population included snack chips, baked products, and corn preparations. We also observed a direct correlation between dietary γ-tocopherol and trans fats (the crude correlation was ~0.76 for all 3 assessment methods; $P < 0.0001$). Some researchers have speculated that γ-tocopherol may be a marker of nutrition-related risk for obesity (45) and trans fat intakes (47). However, mechanisms for these are not clear and warrant further research.

In the current study, δ-tocopherol intake was not associated with its corresponding measure in serum. Vitamin E supplement use (only for the short FFQ) was associated with lower, and serum cholesterol concentration with higher, serum δ-tocopherol concentration. We are not aware of other studies that have specifically examined the determinants of serum δ-tocopherol or its role in disease prevention, but further investigation is warranted.

Food sources of tocopherols. Dietary sources of α-tocopherol in the current study included snack chips, oils, salad dressing, and fish preparations. This is similar to previous observations (17) in a geographically similar population located in the LMD region. Main food contributors in the NHANES (1999–2000) data were tomato products such as spaghetti sauce, pizza, and chili; potato chips; salad dressings; and baked sweets. γ- and δ-Tocopherol sources in the current study included snack chips, baked desserts, margarine, and corn preparations.

In summary, just over one-half of the DPASS participants met the EAR for α-tocopherol from diet and supplements and ~5% met it from diet alone. The FFQ used in the DPASS and the JHS appear to be useful for the measurement of total α-tocopherol intake. For γ-tocopherol, vitamin E supplement use was negatively associated with serum γ-tocopherol concentrations and may have limited our ability to detect associations between γ-tocopherol intake and serum concentrations. There was a positive association between BMI and serum γ-tocopherol concentrations, which warrants further investigation. Main dietary sources of α-tocopherol were not rich sources of the nutrient but were more frequently consumed.

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